genetic screen for worms with abnormal pharynx muscle morphology facilitated by an integrated *myo-2GFP* reporter gene, allowing for rapid identification of animals with misshapened or missing pharynx. We have focused on two major classes of mutants, those with a short, wide pharynx, and those with amorphously shaped pharynx muscle cells. Twenty mutants manifested short and wide pharynges, suggesting that genes required for embryonic elongation of the pharynx were mutated. We have mapped many of these to small regions of chromosomes by SNP mapping. We have identified that two of the mutant lines represent alleles of sma-1, a beta-spectrin; however, many other mutant lines do not map to any previously described short-pharynx gene loci. Most of these mutant lines are larval lethal; acrylic bead feeding assays have shown that they are unable to ingest food. Further mapping will be required to identify the actual genes resulting in the phenotypes. In addition, we have identified a series of mutant lines in which the pharynx muscle cells do not appear to have normal adhesion to the pharynx. We have mapped two alleles with this phenotype to chromosome I, and shown that they do not complement each other. The acrylic bead feeding assays also suggest that the arrested larvae are not capable of ingesting food. Because of the amorphous shape of the pharynx, we are counting the number of pharynx muscle nuclei present to determine if muscle cells are missing.

doi:10.1016/j.ydbio.2009.05.395

Program/Abstract # 366
Sprouty function in pancreas morphogenesis
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Two epithelial buds from the endoderm undergo a series of morphogenetic events to form the definitive mouse pancreas. To study this, the three dimensional structure of the pancreatic epithelium was examined at various stages of development using a combination of immunostaining and confocal techniques. Examination of several wild-type CD1 specimens collected between embryonic days (E)9 and E14 suggests that pancreas morphogenesis is stereotyped, allowing us to reconstruct the series of morphogenetic events that lead to the formation of the definitive pancreas. Using this system, we have begun to examine the function of Sprouty (*Spry*) genes, which encode feedback antagonists of Receptor Tyrosine Kinase (RTK)-signaling, in pancreas morphogenesis. Removing either *Spry1* or *Spry2* singly has no obvious effect, however removing combinations of these results in defects in the morphogenesis of the pancreatic epithelium. Furthermore, ectopic budding of the endoderm is observed near the region where the pancreas normally forms. The significance of these phenotypes is currently being investigated.

doi:10.1016/j.ydbio.2009.05.396

Program/Abstract # 367
ENU screen for mutations in zebrafish pancreas development
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A haploid ENU (N-ethyl-N-nitrosourea) screen was performed on Ekkwill (EK) males to look for endocrine pancreas mutations by screening with *islet-1* (*isl1*). We recovered and confirmed in diploids, two pancreas mutants from our screen. 835.4 mutant embryos lack *isl1* expression in the endoderm but maintain CNS expression. Also, they lose all pancreas, liver and intestinal markers. It was found that they lose early endoderm markers such as *sox17*, *foxa2*, and *foxa1*, but have decreased *sox32* expression. As they develop, they become curved and develop a swollen pericardium by 48hpf and is embryonic lethal by day 4/5. We are currently mapping the 835.4 mutation by using CA repeat markers. 88.21 mutant embryos lack *islet-1* expression in the endoderm, but maintain CNS expression. We mapped the mutation to the catalytic domain of the *raldh2* gene where there are already 2 alleles, *neckless* (*nls*) and *no fin*. Our phenotype is similar to *nls*, in that no fin buds develop, swollen pericardium appears 3dpf and is embryonic lethal by day 4/5. Like *nls*, 88.21 mutants lose many endoderm markers, but upon further evaluation it was discovered that both *nls* and 88.21 maintain some endoderm marker expression. We injected a translational morpholino targeting *raldh2* and were able to knockdown endoderm marker expression. When we treat wildtype embryos with DEAB, an inhibitor of retinaldehyde dehydrogenases, endoderm marker expression is completely abolished. We performed qPCR and low levels of maternal *raldh2* mRNA was detected at 3hpf which may explain the residual expression seen in *nls* and 88.21.

doi:10.1016/j.ydbio.2009.05.397

Program/Abstract # 368
Withdrawn

doi:10.1016/j.ydbio.2009.05.398

Program/Abstract # 369
Role of tight junction proteins in zebrafish liver morphogenesis
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Hepatocytes are the primary cells in the liver that produce bile for lipid metabolism. During liver morphogenesis, hepatocyte precursors are actively migrating, proliferating, and differentiating into hepatocytes or cholangiocytes (epithelial cells of the bile ducts). Bile duct formation also requires the polarization of hepatocytes such that their apical domain defines the bile ducts. This apical domain is defined by tight junctions. In an *in situ* hybridization expression screen in zebrafish, a member of the claudin family was found to be expressed primarily in the liver in the early stages of liver morphogenesis. Its expression becomes restricted to specialized cells of the liver at later stages. Claudins are membrane proteins that are part of the tight junctions. Their main function is to act as a paracellular transport barrier. Surprisingly, knockdown of claudin expression causes a small liver phenotype, suggesting that it plays a role in early liver morphogenesis. The role of Claudins in migrating, proliferating, and differentiating cells is unknown. They were initially identified as calcium-independent adhesion molecules mediating cellcell contacts. During early stages of liver morphogenesis, hepatic cells migrate out of the intestinal rod as a cohort of cells. One possible non-canonical role of Claudins may be to act as adhesion molecules to facilitate migration. Furthermore, recent *in vitro* evidence suggests that tight junctions communicate with the nucleus to mediate proliferation, gene expression, and differentiation depending on the cellular environment. My project aims to investigate these non-canonical roles of claudins during liver morphogenesis.

doi:10.1016/j.ydbio.2009.05.399